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anthracene with (plant or yeast)

32

L1

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L6 ANSWER 27 OF 27 CAPLUS COPYRIGHT 2002 ACS

AN 1948:32360 CAPLUS

DN 42:32360

OREF 42:6893b-g

TI The enzymic hydrolysis of hydrogen peroxide in **plant** extracts.  
Genetic and chemical influence on the enzyme formation

AU v. Euler, Hans

SO Arkiv Kemi, Mineral., Geol. (1946), 24A(No. 13), 15 pp.

DT Journal

LA German

AB cf. C.A. 24, 1872. The enzymes which contribute to the synthesis of chlorophyll as well as to the catalase-hemins are influenced by inhibitors whose formation are genetically detd; Chlorophyll-defective mutants can be produced by x-ray irradiation. Diminishing catalase activity in chlorophyll-defective plants was investigated using barley-leaf exts. The leaves were ground and suspended in a pH 7.3 buffer and used as such for catalase detn. The av. reaction catalase const. (k) for 50 mg. normal, 10-days old barley seedlings was 0.083. The k for chlorophyll-defective seedlings was 0.015. By adding a heated ext. of seedlings to the reaction mixt. no inhibitory effect was observed. The k is not affected by inhibitors; the difference between k values of normal and chlorophyll-defective plants is not due to inhibitors. The genes responsible for the formation of the porphyrins must then be inactivated. It is indicated that these mutations are related to the nucleoprotein-enzyme systems. If the seeds are soaked in 25 or 40% heavy water for 25 hrs. prior to germination, it is observed that D delays the germination of the seeds. (Bonhoefer, et al., C.A. 30, 7131.1). The 40% soln. has a greater effect than the 25% soln. and the delay is greatest immediately after swelling which means that the effect is felt only as long as D is present in the seed as a reserve material or its immediate metabolites. It may be concluded that D has no lasting effect either on the structure or on the activity of the enzymes. The k of green leaves of D-treated seeds is after 6-days germination (30 mg.) 0.0122 and for H2O-treated seeds 0.0151. After 22-days germination k (30 mg.) is 0.0108 and 0.0124, resp. It was found impossible to differentiate between catalase activity of diploid and triploid plants (aspen). If pollen of a rye **plant** is chemically treated and the seeds developed from such plants are allowed to germinate, no clearcut picture is obtained as to leaf size or catalase activity. During a chem. treatment of pollen part of it is not attacked and behaves as normal pollen and part is affected and cannot cause any fertilization. The later development of such seedlings show great differences in leaf shape, which may be caused by **mutation**. Particularly effective in this respect was camphor, **anthracene**, and benzoquinone. In plants treated with these no ears of grain were developed.

L6 ANSWER 26 OF 27 CAPLUS COPYRIGHT 2002 ACS

AN 1971:84338 CAPLUS

DN 74:84338

TI Dithranol and dithranol-like compounds. II. Mutagenicity

AU Zetterberg, Gosta; Swanbeck, Gunnar

CS Inst. Physiol. Bot., Univ. Uppsala, Uppsala, Swed.

SO Acta Dermato-Venereol. (1971), 51(1), 45-9

CODEN: ADVEA4

DT Journal

LA English

AB Of 11 **anthracene** and anthraquinone derivs., dithranol (I) was the most effective in inducing respiration-deficient (RD) mutants in *Saccharomyces cerevisiae*. 1,9-Dihydroxyanthracene and 1,8,9-trihydroxy-3-methylanthracene also induced such mutants but to a lesser extent than I. After prolonged use, 1,8-dihydroxyanthraquinone (II) and 1,8-diacetoxyanthraquinone also induced a low but significant increase in the proportion of RD mutants. Compds. which induced RD mutants were all strong DNA binders in vitro. Anthraquinone derivs. were not as effective as anthracenes in RD induction, although many were strong DNA binders in vitro. The frequency of chromosomal mutations was not increased by treatment of **yeast** or *Ophiostoma* with I. The mechanism for the induction the RD **mutation** with these compds. is discussed and a hypothesis is presented to explain the antipsoriatic effect of I.

L6 ANSWER 24 OF 27 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.  
 AN 79251624 EMBASE  
 DN 1979251624  
 TI Toxic and mutagenic effects of carcinogens on the mitochondria of  
 Saccharomyces cerevisiae.  
 AU Egilsson V.; Evans I.H.; Wilkie D.  
 CS Dept. Bot. Microbiol., Univ. Coll. London, London WC1 6BT, United Kingdom  
 SO Molecular and General Genetics, (1979) 174/1 (39-46).  
 CODEN: MGGEAE  
 CY Germany  
 DT Journal  
 FS 037 Drug Literature Index  
 016 Cancer  
 030 Pharmacology  
 004 Microbiology  
 LA English  
 AB Nineteen haploid **yeast** (*Saccharomyces cerevisiae*) strains were  
 used to assess the relative growth inhibitory potencies on fermentable vs.  
 non-fermentable media of a collection of carcinogenic and non-carcinogenic  
 chemicals. The majority of carcinogens were distinctly more potent on the  
 non-fermentable (glycerol) medium, where mitochondrial function is  
 required for growth, than on the fermentable medium, where it is not. The  
 anti-mitochondrial selectivity indicated by these growth tests was much  
 slighter for the non-carcinogens. Similarly most carcinogens induced the  
 cytoplasmic petite **mutation** whereas the non-carcinogens did not.  
 Five carcinogens which were tested impaired the development of cytochromes  
 aa(3) and b in glucose cultures. Six carcinogens, when tested, inhibited  
 growth on three fermentable sugars, the utilisation of which requires  
 mitochondrial function. Out of five carcinogens which were examined, four  
 suppressed the surface-dependent phenomenon of flocculence in a  
 flocculating strain of **yeast**, at concentrations primarily  
 affecting the mitochondrial system; the fifth had a similar but less  
 pronounced effect.

S

(FILE 'HOME' ENTERED AT 16:47:14 ON 08 AUG 2002)

FILE 'MEDLINE, CANCERLIT, BIOTECHDS, CAPLUS, EMBASE, BIOSIS' ENTERED AT  
16:47:30 ON 08 AUG 2002

L1	72829 S ANTHRACENE
L2	1253541 S HYPERMUTA? OR MUTATION OR MISMATCH REPAIR OR PHENOTYP?
L3	1769216 S PLANT OR YEAST
L4	1933 S L3 AND L1
L5	52 S L4 AND L2
L6	27 DUP REM L5 (25 DUPLICATES REMOVED)
L7	1580749 S ASSAY OR PCR
L8	115 S L7 AND L4
L9	71 DUP REM L8 (44 DUPLICATES REMOVED)
L10	54 S L9 AND PLANT
L11	1 S L10 AND PCR
L12	10 S L10 AND (DNA OR NUCLEIC OR GENE)

L6 ANSWER 17 OF 27 MEDLINE  
 AN 88225041 MEDLINE  
 DN 88225041 PubMed ID: 3286249  
 TI Induction of mitotic chromosome loss in the diploid **yeast**  
 Saccharomyces cerevisiae D61.M by genotoxic carcinogens and tumor  
 promoters.  
 AU Albertini S; Friederich U; Wurgler F E  
 CS Institute of Toxicology, Swiss Federal Institute of Technology,  
 Schwerzenbach ZH.  
 SO ENVIRONMENTAL AND MOLECULAR MUTAGENESIS, (1988) 11 (4) 497-508.  
 Journal code: 8800109. ISSN: 0893-6692.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 198806  
 ED Entered STN: 19900308  
 Last Updated on STN: 19900308  
 Entered Medline: 19880628  
 AB Three genotoxic carcinogens and eight tumor promoters were tested for  
 induction of aneuploidy, specifically chromosome loss, in Saccharomyces  
 cerevisiae D61.M. This is a heterozygous diploid **yeast** strain  
 that permits the scoring of segregants expressing three linked recessive  
 markers (cyhR2, ade6, and leu1), two of which (ade6 and leu1) are located  
 close to the centromere on opposite arms of chromosome VII. The centromere  
 marker leu was routinely checked, and a positive control (bavistan) was  
 run with every experiment. The three genotoxic carcinogens aflatoxin B1,  
 benzo(a)pyrene, and 7,12-dimethylbenz(a)**anthracene** did not  
 induce aneuploidy, independent of the presence or absence of an exogenous  
 metabolic activation system (rat liver homogenate; S9). Four of the eight  
 tumor promoters tested induced chromosome loss but not mitotic  
 recombination or **mutation**: cholic acid, lithocholic acid,  
 phenobarbital, and saccharin. Diethylstilbestrol (DES) led to positive as  
 well as to negative results in several independent experiments. In the  
 case of the positive experiment, DES also induced putative recombinants.  
 Three tumor promoters induced neither chromosome loss nor mitotic  
 recombination: anthralin, 4,4'-dichloro-diphenyl-ethane (DDT) and  
 gamma-hexachlorocyclohexane (lindane). From our experiments it can be  
 concluded that the hypothesis put forward by Parry et al. [Nature;  
 294:263-265], according to which tumor promoters induce chromosome loss in  
**yeast**, is not correct in a general sense. In our set of eight  
 tumor promoters, only one half distinctly induced chromosome loss.

L6 ANSWER 1 OF 27 CAPLUS COPYRIGHT 2002 ACS

AN 2001:753502 CAPLUS

DN 136:324416

TI Anti-mutagenicity of **plant** origin lactic acid bacteria isolated from rice and processed rice products

AU Kumagai, Takehisa; Seno, Kimiko; Watanabe, Toshiyuki; Okada, Sanae

CS Kameda Seika Co., Ltd., Kamedamachi, Nakakanbara-gun, Niigata, 950-0192, Japan

SO Nippon Shokuhin Kagaku Kogaku Kaishi (2001), 48(9), 693-696  
CODEN: NSKKEF; ISSN: 1341-027X

PB Nippon Shokuhin Kagaku Kogakkai

DT Journal

LA Japanese

AB Nine strains of **plant** origin lactic acid bacteria were isolated from rice and processed rice products to exam. their anti-mutagenic effect. Tested Lactic acid bacteria were six strains of *Lactobacillus casei* subsp. *casei* and three strains of *L. plantarum*. Used mutagens were 3-amino-1,4-dimethyl-5H-pyrido-(4,3-b)-indole (Trp-P1), 3-1,4-dimethyl-5H-pyrido-(4,3-b)-indole (Trp P2), 2-amino-**anthracene** (2-AA), and N-methyl-N'-nitro-N-nitrosoguanidine (MNNG). Anti-mutagenic activity on living and killed cells of lactic acid bacteria were measured using *Escherichia coli* WP2uvrA. All strains showed anti-mutagenic activities against Trp-P1, Trp-P2 and 2-AA. The activity against Trp-P1 was higher than the other mutagens. Living and killed cells of lactic acid bacteria showed similar activity against Trp-P1 and Trp-P2. Four strains of killed cells of *L. casei* subsp. *casei* had higher activity than the living cell against 2-AA. Only living cells of *L. casei* subsp. *casei* had anti-mutagenic activities against MNNG. This result was similar to previous report using 4-nitroquinoline-1-oxide.